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A CLINICAL STUDY OF DIABETIC FOOT ULCERS

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CERTIFICATE

This is to certify that the dissertation entitled **"A CLINICAL STUDY OF DIABETIC FOOT ULCERS"** submitted by **Dr. S. MUTHURAJ** to the faculty of General Surgery, Government Rajaji Hospital, Madurai Medical College, Madurai, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, is in partial fulfillment of requirement in the award of M.S. Degree, branch – I (General Surgery) for the September 2006 examination is a bonafide research work carried out by him under my direct supervision and guidance.

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CONTENTS

DECLARATION

I solemnly declare that this dissertation **"A CLINICAL STUDY OF DIABETIC FOOT ULCERS"** was prepared by me from the Department of General Surgery, Madurai Medical College and Govt Rajaji Hospital , Madurai , under the guidance and supervision of **Prof. Dr.S. Vijayalakshmi M.S., Professor of Operative Surgery, Madurai Medical College , Madurai.**

This dissertation is submitted to the Tamil Nadu Dr.M.G. R. Medical University, Chennai ,in partial fulfillment of the university regulations for the award of the degree of M.S. Branch I General Surgery examinations to be held in September 2006.

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INTRODUCTION

Diabetes is a global problem. The majority of diabetic develop foot ulcers in one point of time or other during the course of their lifetime. It can be aptly called as the ‘Modern day Leprosy’. A significant number of such patients will require amputations. The etiopathogenesis of diabetic foot lesions are multifactorial. Diabetic neuropathy, vasculopathy, poor control of diabetes and bacterial infection are some of them.

"Every other diabetic is a surgical diabetes" – Joslin

The reasons for prevalence of foot ulcers in diabetes;

1. Foot is the most vulnerable part of the body for injury and infection and neglected by the patient.
2. The site of preference for neuropathy and ischaemia is also the foot.

India ranks first in the world in the prevalence of diabetes cases. Foot ulcers develop in approximately 15 percent of patients with diabetes, and foot disorders are a leading cause of hospitalization among such patients. Eighty-five percent of lower-limb amputations in patients with diabetes are preceded by foot ulceration, suggesting that prevention and appropriate management of foot lesions are of paramount importance. The complications are more prevalent among the people of lower economic status due to negligence, illiteracy and poverty and sedentary lifestyle.

AIMS OF THE STUDY

The aims of the study are;

1. To understand the pathology of diabetic foot ulcers and relative distribution of this condition among diabetic foot patients admitted in Government Rajaji Hospital from August 2003 to February 2005.
2. To study the mode of presentation of diabetic foot ulcers and their risk factors.
3. To study the benefit and outcome of the different treatment modalities for diabetic foot which are practiced in our institution.
4. To assess the socioeconomic factors and their implications.

REVIEW OF LITERATURE

HISTORY OF DIABETES MELLITUS

Diabetes is an age old disease affecting about one percent of the population equally in males and females.

The history of diabetes[31,33] is briefly given

Charaka 2nd century AD in his "Charaka Samhita" has mentioned the sweetness of urine in addition of the symptom of polyuria. The Egyptian Medical Journal "Papyrus Ebers" (1500 BC) which was discovered at Luxor in Egypt in 1872, has recorded the symptoms of passing of frequent and large quantities of urine. Avicenna (980 – 1037) an Arab physician gave the first description of diabetic gangrene.

Thomas Cawly (1778) noticed abnormal changes in pancreas, in fatal cases of diabetes. Cullen 1709 – 1790) added the adjectives mellitus to the disease in order to distinguish from diabetes insipidus.

The sugar in diabetic urine is glucose was first shown in 1815 by Micael Eugene Chevruel. The first suggestion that plantar ulceration might be due to neurological deficit was made by Duplay and Morat in 1873

A German medical student Paul Langerhans noted that the pancreas contain two distinct groups of cells – the acinar cells, secrete digestive

enzymes and cells that are clustered in islands which he suggested served a second function.

Fehling reported his quantitative test for sugar in the urine in 1850. In 1908 Benedict' introduced Benedict's test for urinary sugar in diabetes.

A young Canadian orthopedic surgeon by name Fredrick Banting was interested in diabetes, along with a young second year medical student by name Charles H. Best, in the laboratory of Prof. J.J.R. Macleod in Toronto, and discovered the insulin (1922- 1936). The discovery revolutionized the treatment of diabetes and its complications.

The diagnosis of insulin in the 1920s resulted in increased lifespan of the diabetics and ultimately with increased complications. The edipemic of diabetic foot disorders started in 1940 -1950 and is still continuing. Diabetes mellitus is the leading cause of non traumatic lower extremity amputation in the United States. With the growing number of patients with amputations and chronic ulcers

diabetic foot disease can be called "Modern day Leprosy".

SURGICAL ANATOMY OF FOOT[38]

SKIN AND NAILS

The skin of dorsum of the foot is thin and highly flexible, containing hair follicles, sweat glands and scanty sebaceous gland. Hair are sparse and thick. It is less than 2 mm thick and few fibrous septa penetrate to deeper fascial structures. The plantar skin is 5 mm thick especially over those points which bear weight viz, heel, and ball of big toe and lateral margins of the sole. It has no hair follicles or sebaceous glands but sweat glands are numerous.

Hypodermis is not part of the skin but attaches the skin to underlying structures. Hypodermis is composed of loose areolar connective tissue most of this collagenous and elastic fibres running parallel to the surface of the skin, but some are continuous with the fibres of dermis. Often fat cells are

deposited among these fibres; hypodermis is well supplied with blood vessels and nerve endings. Tactile sensations are exceptionally good in the sole.

The subcutaneous tissue in the sole as in the palm differs from that of the rest of the body in being more fibrous, tough and stingy. Fibrous septa divide the tissue into small loculi which are filled with fluid fat under tension; this makes a shock absorbing pad especially over the heel and over the tips of toes.

Deep fascia of the dorsum of the foot (fascia dorsalis pedis) is the thin layer continuous above with the inferior extensor retinaculum at the sides of the foot, it blends with plantar aponeurosis anteriorly it ensheaths the dorsal tendons.

Plantar aponeurosis covers the whole length of the sole. It arises posteriorly from the medial and lateral tubercles of the calcaneous and from the back of that bone below the insertion of the tendocalcaneous. It spreads out over the sole and is inserted by five slips into each of the five toes. It lies superficial to the vessels. The muscles and tendons and consists of 3 portions, relatively thin medial and lateral parts, a very dense and strong intermediate part.

NERVES

Cutaneous nerves are arranged in the following way. The medial plantar nerve supplies the three and a half digits on the medial side of the

foot. The lateral plantar nerve supplies lateral one and half digits. The medial calcaneal branches of the posterior tibial nerve supply the skin under the heel.

The motor and sensory components of the sciatic nerve supply the foot. The innervations to the sole are from medial calcaneal branch of tibial nerve.

VASCULATURE OF THE FOOT

All the arterial supply of the foot is derived from popliteal artery. The anterior tibial artery enters the extensor compartment of the leg and becomes dorsalis pedis artery in the foot. The posterior tibial artery divides into medial and lateral plantar arteries. The medial plantar artery runs forward on the medial side of the medial plantar nerve. The artery supplies medial side of the foot and its digital supply is restricted practically to the big toe. Lateral plantar artery crossed the sole crosses the sole obliquely on the marginal side of the nerve, just deep to the first layer of the sole toward the base of the fifth metatarsal bone. their plantar arch curves convexly forward across the bases of fourth, third and second metatarsals and is joined in the proximal part of the first inter metatarsal space by the dorsalis pedis artery from the convexity of the plantar arch, plantar metatarsal arteries run forward and bifurcate to supply the four web space and digit. The veins accompany the perforating

arteries take most of the blood from the sole and from the interosseous muscles to the dorsal venous arch.

MUSCLES OF FOOT

The muscles in the extensor group are located anteriorly in the leg, they include tibialis anterior, extensor hallucis longus, laterally are the peroneal muscles. The flexors are in the posterior compartment of the leg. The deep fascia encloses the muscles in the leg. In the sole of the foot the plantar aponeurosis is the most superficial layer the fibres of plantar fascia is divided into five processes. Beneath the fascia the muscles in the sole of the foot are categorised into four layers only the muscles of first layer cover the whole extent of the foot

BONES OF THE FOOT

The bones of the foot are the tarsal bones, metatarsals and the phalanges. The tarsal bones are the calcaneum the talus, the navicular, the cuboid and the three cuneiform bones. Calcaneum is the largest bone of the foot and forms the prominence of the heel it articulates with Talus above and Cuboid in front.

Talus carries the whole body weight. It lies on weight bearing calcaneum below the tibia and communicates thrust from one to the other. Navicular bone can be seen and felt on the medial border of foot. Cuboid bone is rather wedge shaped and articulates with lateral cuneiform.

Cuneiform bones all three are wedge shaped. All the three completes medial longitudinal arch. The metatarsal bones and phalanges resemble metacarpals and phalanges of hand. Each toe has three phalanges except the big toe which possess only two.

ETIOPATHOGENESIS OF DIABETIC FOOT

In United States there are 11 million diabetics with a total of 110 million diabetic toes[26]. Compounding the diabetic foot are problems like peripheral vascular disease, neuropathy and overweight. These lead to ulceration and amputation. 15 percent of diabetic will develop diabetic foot ulcers. Amputation rates are 15 times more common in diabetics than non diabetics.

MECHANISM OF DEVELOPMENT OF FOOT LESIONS

The factors leading to diabetic foot can be classified as follows[32]

1. *Predisposing factors:*

    Vascular disease

    Neuropathy

    Liability to infections

2. *Precipitating factors:*

Physical injury

Mechanical trauma

Heat

3. *Aggravating factors:*

Infection

Ischaemia

Neuropathy

VASCULAR DISEASE

Disease of blood vessels is the major cause of complication of diabetic foot. It may affect capillaries, the smallest blood vessels and the aorta[33].

ATHEROSCLEROSIS

It may be defined as a degenerative vascular disease composed of fibrous and or fatty change affecting principally the tunica intima of arteries. Atherosclerosis in lower limbs of type II diabetic patients was 20 times more common than age and sex matched controls.

PATHOLOGY

The process first affects innermost layer of wall, the tunica intima, where accumulation of lipid filled cells occurs. As the process extends tunica

media becomes involved. Vascular smooth muscles cells proliferate and fibrosis increases. Progression of disease will lead to destruction of elastic wall. Finally intimal plaque may necrose resulting in discharge of atheromatous material leading to embolism, ulceration and thrombosis.

Abnormalities of blood lipids are shown in about half of the patients. Usually concentration of low density lipoprotein is elevated. In type II diabetic patients HDL is usually reduced in concentration. HDL protects against atherosclerosis and hence low HDL concentration will lead to atherogenesis. Other abnormalities being change in VLDL, LDL, lipoproteins and change in the proteins (apoproteins)

There is increased predisposition for systolic hypertension in diabetic patients. In type I diabetes there is also evidence of diastolic hypertension due to coexistent diabetic nehpropathy. Hyperglycaemia causes impairment of vessel wall nutrition, changes in coagulation, or osmolar effect. Hypercoagulation state is produced by several mechanisms in diabetic patients. Cigarette smoking aggravates atherogenesis[35]

MORPHOLOGY AND DISTRIBUTION

Although atherogenesis[34] occurs in diabetic and non diabetics, the distribution varies. In diabetic patients commonest angiographic patterns are Aorta and iliac arteries, common and superficial femoral arteries are usually patent. Profunda femoris is the most severely and irregularly damaged in

distal part. Proximal part of femoral artery is often patent but distal part is occluded. The tibial arteries are severely diseased. There are no identifiable named calf arteries or only one of the major arteries is seen[27]. The major pedal vessels are not severely affected.

From the point of view of treatment most serious feature of this pattern is occlusion of distal popliteal arteries and its branches which is difficult to correct surgically.

CALCIFICATION OF ARTERIES

Calcification of tunica media of muscular arteries is very common in diabetes. It is seen that calcification is associated with peripheral neuropathy rather than angiopathy. Calcification has been used as prognostic indicator in diabetes. Calcification usually limited to tunica media. Intima is usually unaffected. Thus resting perfusion is not compromised.

MICROANGIOPATHY

Two major causes of morbidity in diabetes are nehpropathy and retinopathy has been known for many years to be due to microvascular disease. These changes are divided in 2 groups.

1. ***Thickening of capillary basement membrane.***

2. ***Proliferative changes in arterioles and arteries. which may cause impairment of flow and affects the availability of oxygen in the tissues, the blood flow.***

NEUROPATHY

Impairment of nerve function is an important and frequent complication of diabetes. This predisposes to diabetic foot[24].

PATHOLOGY

Essential pathology in diabetic neuropathy is nerve fiber loss. Findings include segmental demyelination, hypertrophy of Schwann cells and perineural fibroblasts[29,32].

ETIOLOGY

Hyperglycemia and consequent metabolic derangement like sorbitol and fructose accumulation. interneural myoinositol deficiency, non enzymatic glycosylation of nerve protein and impaired axoplasmic transport may play an important role in development of diabetic neuropathy. There is also increasing evidence of vascular component in the aetiology of diabetic peripheral neuropathy.

SENSORY CHANGES

Most significant abnormality in the development of foot lesions is the inability to perceive pain. This predisposes foot to mechanical injuries. Injury as well infection goes unnoticed. Neurogenic inflammation is also reduced.

MOTOR CHANGES

Denervation of muscles has important effects on the functions of foot. The small muscles of foot affected. Due to denervation metatarsophalangeal joints are hyperextended and interphalangeal joints are flexed. The joints initially remain mobile but later degenerative changes occur and joint becomes fixed. Deformities predispose to ulcers due to unequal distribution of pressure

AUTONOMIC CHANGES

Autonomic neuropathy contributes to pathogenesis of ulceration, neuropathic edema and Charcot's arthropathy. Quantitative impairment of sweating and blood distribution has been measured in these conditions.

Impaired sweating contributes to formation of hypertrophied plaques and fissures through dehydration. Sympathetic neuropathy will cause some effects on peripheral circulation by increasing vasodilation and diminished vascular demineralization and rarefaction of bones, which in turn predisposes to recurrent minor fractures resulting in Charcot's arthropathy.

INFECTION AND WOUND HEALING

Increased liability to wound infections in diabetes became part of conventional custom. Diabetic are prone for not only foot infection but also skin infections, osteomyelitis, fungal infection and tuberculosis[27,33].

MECHANISM

Diabetes with infection will have impaired inflammatory and wound healing process by reduced

1. ***Blood supply to affected area due to microvascular disease or macrovascular disease.***
2. ***The effectiveness of inflammatory response. Poor cellular infiltrate, impaired function of phagocytic cells are important features in diabetics.***
3. ***The repair process which results in formation of fibrous tissue. The reason for this being lack of insulin which leads to reduction in levels of collagen. It has been also demonstrated that insulin therapy starting in early post operative period or earlier causes adequate collagen to be deposited in wound***

BACTERIOLOGY

In vitro high concentration of glucose is believed to favour multiplication of bacteria especially gram positive bacteria, e.g.:

staphylococci, streptococci, pseudomonas and gram negative bacteria e.g. Klebsiella, E. coli and proteus.

Diabetic foot is prone for fungal infection as well. Deep infections of the foot are difficult to manage. On an average there are more than three organisms in each infected foot. Rare but lethal infection occurs as a result of anaerobic infections[32].

MECHANICAL FACTORS

The physical factors also play a role in development of foot ulceration and its delayed healing. They may act by

1. ***Disrupting tissue***
2. ***Pressure causing ischemia***
3. ***Repetitive stress causing necrosis.***

MECHANISM OF FOOT INSTABILITY

When man assumed erect posture the foot became subject to a variety of stress which are not encountered by his arboreal ancestors.

ANATOMICAL FACTORS

The longitudinal and transverse arches are very important in weight bearing. Anterior attachment of longitudinal ligament of plantar aspect of foot is in the digits. So when digits are dorsiflexed the ligaments get tensed and this causes the pillars of the arches being brought closer.

CHANGES IN DIABETICS

Sites of ulcer always correspond to the area of the fore foot that carries highest load. Especially in a partially amputated foot or deformed foot due to neuropathic joints the distribution of pressure is unequal. Also maximum load is directly proportional to body weight so that heavier patients run greater risk of developing foot ulcers. Also maximum longitudinal shearing forces occur in the sites of healed ulcers.

CHANGES IN CONNECTIVE TISSUE

Process of non-enzymatic glycosylation by which glucose molecules attach to variety of protein is now known to be widespread in diabetics. Methylated keratin and abnormal inflexible collagen are among the abnormal proteins.

DEVELOPMENT OF NEUROPATHIC ULCERATION

Due to several reasons one or two areas of fore foot in the region of metatarsal heads or interphlalangeal joints of the greater toe come to carry greater than normal loads. Diabetic neuropathy causes no pain and patient is unaware of inflammation. Subsequently an ulcer develops.

CLINICAL PRESENTATION

Clinical presentation of diabetic foot includes signs and symptoms of vascular insufficiency, neuropathy, ulcers, gangrene and infection.

SIGNS AND SYMPTOMS
A. VASCULAR INSUFFICIENCY

- ***Intermittent claudication***
- ***Cold feet.***
- ***Nocturnal pain***
- ***Rest pain***
- ***Absent pulses***
- ***Blanching on elevation***
- ***Dependent rubor.***
- ***Atrophy of subcutaneous fatty tissues***
- ***Shiny skin***
- ***Loss of hair on foot and toes***
- ***Thickened nails***
- ***Gangrene***
- ***Miscellaneous***

    Blue toe syndrome

    Acute vascular occlusion characterized by

- ***Pain, pallor, paraesthesia, pulselessness below the block and paralysis***

B. NEUROPATHY

The signs and symptoms of neuropathy in the diabetic foot and leg include

- ***Parasthesia***
- ***Hyperasthesia***
- ***Hypoasthesia***
- ***Radicular pain***
- ***Loss of vibratory and position sense***
- ***Anhydrosis***
- ***Heavy callus formation over pressure points***
- ***Trophic ulcers***
- ***Radiographic changes like demineralisation***
- ***Osteolysis***
- ***Charcot's joint***

CLINICAL FEATURES

Patient may present with the following features.

Paronychia – Though less common on the feet than on the hands can also occur mainly involving greater toe may be bacterial or candidial which present in web space. Patient present from asymptomatic fungal infection of nail or low grade paronychia.

Several types of major and minor infections occurring in foot are

MINOR INFECTIONS

- ***Fissure in web space.***
- ***Skin thickening diabetic sclerosis.***
- ***Diabetic bullae.***

MAJOR INFECTIONS

CELLULITIS:

Patients presents with cellulitis of the foot involving distal half or whole of the foot because of necrotizing skin and subcutaneous infection these patients manifests with edema involving dorsum of the foot with shiny skin and warmth.

ABSCESS:

Abscess may be localized to single toe or multiple toes or in the deep spaces of the sole. The most important signs are swelling and redness which can be seen on the dorsum of web spaces of the foot. However the most characteristic sign is separation of the toes due to diffuse edema of the deep tissues of the foot and always indicates that there is pus deep in the foot.

ULCER:

Ulcer may present on the dorsal or plantar aspect of the foot. Plantar ulcer also called as trophic or penetrating ulcer and mal performs. There are

typically painless and occur over areas that normally carry weight. The earliest change is an area of hyperkeratosis often over a metatarsal head. As the ulcer extend as a result of infection, spread along the tendon sheaths or other planes opened up by the extension of ulcer. The commonest site of an ulcer of the toe is on the dorsum of the proximal interphalangeal joint of a clawed toe.

GANGRENE:

Areas of gangrene may occur on parts of the foot that are exposed to pressure. The common sites are heel, malleoli and the areas of the first metatarsal head medially, and the base of the fifth metatarsal head. Small areas of gangrene may also occur on the parts of the foot not subject to pressure because of embolism of atheromatous debris. Gangrenous patches may form in the interdigital clefts. If the infection follows it may spread through tissue which because of poor blood supply is unable to confine the process so that further necrosis occur (wet gangrene). In the diabetic usually the wet gangrene occurs. Gangrene may be localized to single toe or multiple toes or extend to whole of the foot.

OSTEOMYELITIS

No consensus exists on the optimal criteria for diagnosing osteomyelitis, but up to two thirds of diabetic patients with foot ulcers may have osteomyelitis. The findings on plain X rays are often suggestive of osteomyelitis (manifested as bone destruction or periosteal reaction, especially

as compared with findings on prior films). Histologic evaluation and culture of a bone specimen are regarded as the gold standard, although differences in outcome that are based on this approach remain to be established. Magnetic resonance imaging (MRI) is now considered the imaging test of choice when osteomyelitis is suspected; the sensitivity and specificity of MRI for osteomyelitis in diabetic patients are 90 percent or greater.

INVESTIGATIONS

The following investigations are done for the diagnosis and treatment of diabetic foot.

1. To demonstrate the extent and severity of the disease process.
2. To screen them for peripheral vascular insufficiency
3. To confirm and control the undercurrent disease interfering with the healing process.

CLINICAL EXAMINATION

All patients admitted are thoroughly assessed in the bedside. All peripheral pulses are checked and sensory examination done in bedside.

A simple method can identify patients who have lost protective sensation. A nylon monofilament (designated 5.07) is pressed against the skin to the point of buckling. Patients who cannot feel the monofilament are at risk for ulceration and require special care. A test with a monofilament is as effective as

more time-consuming tests of vibration and thermal sensation in identifying patients prone to ulceration. All patients with diabetes should be assessed annually with this inexpensive, rapidly performed test.

URINE EXAMINATION

Urine albumin

Sugar

Ketone:

BLOOD EXAMINATION

- *FASTING BLOOD SUGAR:* Fasting blood sugar more than 120 mgm% is indicative of diabetes.
- *POST PRANDIAL BLOOD SUGAR :* After 100 grams of glucose venous blood is checked for glucose level every half for two hours if it exceeds 180 mg % is indicative of diabetes mellitus.
- *ORAL GLUCOSE TOLERANCE TEST:* Sample of blood is taken prior to the tests 100gm of glucose in water is administered rally to an overnight fasting patient.

In normal subject fasting blood sugar is 80 to 120 mgm % and peak of the curve is not above 180 mgm % The blood sugar value return to normal fasting levels are slightly lower at the end of the two hours ant there is no sugar in any sample of urine.

Cholesterol and triglycerides are usually raised. It can be assessed chemically or by electrophoresis.

CULTURE AND SENSITIVITY TESTS

Pus from infected area is cultured for microorganism and their sensitivity to various antibiotics is tested so that appropriate antibiotic can be administered to control the infection. If facilities are available samples should be taken for anaerobic organisms also.

X RAY[37]

X ray of the foot should be taken if there is any suspicious infection deep to the foot e.g. abscess or osteomyelitis. The signs which suggest the presence of osteomyelitis are destruction of bone commonly seen at metatarsophalangeal joint or in the interphalangeal joint of the great tow. Sequestrum and subperiosteal new bones formations are common. A small amount of gas in the tissues or in the abscess cavity may be seen. A large amount of subcutaneous gas especially if it extends the leg indicates the presence of a serious anaerobic infection. In severe ischemia there may be generalized osteoporosis in the bone of the foot. In addition calcification of the metatarsal or digital vessel is commonly seen.

NON INVASIVE EVALUATION

The noninvasive techniques assumed an important role in peripheral arterial ischemic disease. They give an accurate assessment of anatomic and

physiologic vascular status. Fall in the ankle pressure may be used as an objective test for assessing and following the course of the disease in patients with obliterative arterial diseases of the lower extremities. The magnitude of the disease in ankle pressure and its time course are rough guide to the extent of the impairment of blood flow after exercise induced hyperemia.

1. TOE PRESSURE

They provide a highly accurate method for determining the success in the healing of an ulcer or in minor amputation. A toe pressure of 20 – 30 mm Hg below which is healing is doubtful.

2. DUPLEX SCANNING WITH ULTRASOUND ANALYSIS (DOPPLER SCAN)

The recorded Doppler is used in two ways

- To measure segmental systolic pressure
- To provide flow velocity wave form pattern for analysis.

This combines B – mode anatomic capabilities of revealing the location and amount of vessel lumen and stenosis can be recorded with Doppler derived velocity recordings.

3. OTHERS

- PHOTOPLETHYSMOGRAPHY
- SEGMENTAL PRESSURE
- WAVEFORM EVALUATION

INVASIVE TECHNIQUES

01 PERCUTANEOUS FEMORAL ANGIOGRAPHY

Anatomic evaluation of the vascular supply to the leg and foot require arteriography. It is indicated only if revascularization is planned. In young patients with vascular insufficiency diagnosis of obstruction can be made when arteriogram show severe diffuse atherosclerotic disease involving the tibial and peroneal arteries. Being invasive it is associated with complications like bleeding haematoma, thrombosis, contrast allergy and renal dysfunction.

02 DIGITAL SUBTRACTION ANGIOGRAPHY

The initial attempt at subtraction angiography involved is energy subtraction. If two beams of different proton energy impinge on tissue, the lower energy beam will be relatively more attenuated by substances having high atomic numbers (ie. bone and iodine) due to photoelectric effect various tissues could therefore be subtracted from final image. This is associated with better resolution and can be done as outpatient basis.

03 RADIONUCLEOTIDE BONE SCINTIGRAPHY:

- Bone scanning using technetium 99 m phosphonates are useful in identifying early osteomyelitis.

04COMPUTED TOMOGRAPHY

Well suited for imaging complex articulations and numerous soft tissue structures it can identify and characterize the extent of soft tissue infection.

05MAGNETIC RESONANCE IMAGING

Detects and displays bone marrow alterations in osteomyelitis. It can displays the contrast between soft tissue, medullary tissue and cortex with clarity

RISK ASSESSMENT OF DIABETIC PATIENT FOR ULCERATION[39]

	Low risk	**Moderate risk**	**High risk**
Sensation	Normal	Neuropathy	Neuropathy
Vision/Mobility	Normal	Normal	Impaired
Vascularity	Palpable pulses	Absent pulses	Absent pulses

History	No previous ulcer	No previous ulcer	Previous ulcer
Appearance	No deformity	No deformity	Deformity

RISK ASSESSMENT

It is useful to stratify the information obtained and grade the degree of risk of ulcer development for individual patients. This helps determine how intensive foot observation has to be and can impress on high risk patients the need for vigilance.

MANAGEMENT OF DIABETIC FOOT

Management of a diabetic patient with foot disorder is a multidisciplinary approach. It includes the surgeon, diabetologist, vascular surgeon, orthopaedician, podiatrician, and the patient also.

CONSERVATIVE MANAGEMENT

Diabetic foot is usually dry due to autonomic neuropathy, this will lead to cracks and maceration and subsequent infection. Hence lubricant such as

petroleum jelly, non scented hand lotions or aqueous lanolin is used. Lubricants should not be used in between toes.

Peripheral neuropathy can rarely cause pain and parasthesia. In such case Carbamazepine or Phenytoin sodium can be used. Vitamins' including B 12 is tried by many. In some patients, tricyclics are used.

SAVING THE DIABETIC FOOT

One of the primary goals of treating diabetics is to save the diabetic foot this can be achieved by

1. Correction of vascular risk factors
2. Improved circulation.
3. Proper treatment of diabetic foot ulcers
4. Team work
5. Patient education on foot care.

CORRECTION OF VASCULAR RISK FACTORS

Risk factors for microvascular disease are given in table below. Certain risk factors can be controlled and hence should be treated.

Non treatable	Treatable
Genetic	Smoking

Age	Hypertension
Diabetes	Hypercholesterolemia
Duration of diabetes	Hypertriglyceridemia
	Hyperglycaemia

IMPROVED CIRCULATION

Exercise is important in building collaterals. Vasodilators have a minimal role as diabetes is not a vasospastic condition. Antiplatelet drugs like aspirin and dipyridamole are used. The basic pathology in blood is hypercoagulability and change in rheologic properties of RBC. The ability of the RBC to change shape is lost to certain degree. Pentoxyphylline is a drug which can increases the red cell flexibility. Thus blood flow can be increased and blood viscosity decreased.

TREATMENT OF DIABETIC FOOT ULCERS

The primary approach in treatment of foot ulcer consists of

1. Evaluation: clinically as radiologically to establish depth presence of osteomyelitis and foreign bodies.

2. Metabolic control: Strict glycaemic control for adequate metabolic control.
3. Culture and sensitivity
4. Antibiotic Therapy: Because all skin wounds harbor microorganisms, swab cultures are not useful in clinically uninfected patients, and infection of ulcers is therefore diagnosed clinically. A commonly accepted definition of foot infection is the presence of systemic signs of infection (e.g., fever, leukocytosis) or purulent secretions, or two or more local symptoms or signs (redness, warmth, induration, pain, or tenderness) Started at once according to the prevailing antibiotic sensitivity for gram positive and gram negative organism (Cefotaxime and Ampicillin gram along with Metronidazole) and later according to the pus culture sensitivity report. Parenteral antibiotic are preferred.
5. Limb-threatening infections require immediate hospitalization and bed rest even if signs and symptoms of systemic infection are absent. Up to one third of patients must still undergo major limb amputation before any treatment is initiated -- a fact that underscores the need for aggressive in-hospital treatment of limb-threatening infections. Although medical stabilization, glycemic control, and antimicrobial therapy are important, debridement should not be delayed. Diabetic

patients do not tolerate undrained suppuration. Failure to debride necrotic, infected tissue and drain purulent collections increases the risk of amputation. Drainage by needle aspiration or percutaneous drains is inadequate. All necrotic and devitalized tissue must be debrided, and dependent drainage established. The initial debridement must be performed independently of the status of the arterial circulation, with revascularization postponed until sepsis is controlled. Adequate debridement may require multiple procedures. If the infection has destroyed the architecture and function of the foot or acutely threatens the patient's life, guillotine amputation to control sepsis, followed by definitive closure, is recommended.

6. Daily debridement.
7. No weight bearing. This is essential in healing of ulcers. Modern contact cast allow patient to bear weight. These casts protect the ulcer, decrease edema, and redistribute pressure. Casts are contraindicated in patients with severe peripheral vascular, obesity, osteomyelitis, ataxia, and blindness and in the aged.
8. Vascular surgery: When ulcer not healing even after intensive treatment, vascular surgery should be considered.
9. Post healing treatment: Patient education is important in management of foot after healing Healed ulcer area is vulnerable to break down.

So patient must be educated to reduce the amount of walking and to take shorter steps. Job change may be needed and also special shoes may be needed.

The foot ulcer therapy includes use of a variety of topical preparations which include proteolytic enzymes, antibacterial such as povidone iodine and resins. Use of hyperbaric oxygen is of doubtful value. But none of the topical agents have been shown to improve healing and povidone iodine and hydrogen peroxide have been shown to delay healing.

TEAM APPROACH

Case of diabetic foot is complicated task and requires the teamwork of variety of medical disciplines, which include podiatrics, orthopedics, physiotherapy, vascular surgery, radiology, infectious diseases expert and nurse educators.

PATIENT EDUCATION

Patient education is important to prevent diabetic foot disease, the advice given to patient include[26,24.]

- Inspect toes and areas between toes daily for blisters, cuts or scratches

- Wash feet daily, dry carefully, especially between toe
- Avoid extreme of temperatures. Test water before bathing
- If feet becomes cold at night wear socks, do not apply hot objects
- Do not use chemical agents to removal of corns
- Inspect inside to shoes daily for foreign objects
- Shoes should be comfortable at the time of purchase
- Do not walk barefoot
- Stop smoking
- Cut nails, do not cut corns or calluses
- See physician regularly and get your feet examined

WAGNER'S GRADING OF FOOT LESIONS[1,2]

Wagner (1983) grades lesion of diabetic foot from 0 – 5 by depth and extent.

- **A Grade 0** No ulcer but high risk foot.
- **B Grade 1** Superficial ulcer with no bony involvement

- **C Grade 2** Deep ulcer with no bony involvement
- **D Grade 3** Abscess with bony involvement
- **E Grade 4** Localized gangrene
- **F Grade 5** Gangrene of the whole foot

The other system is University of Texas system which takes into account of size, depth and presence of absence of infections also has good prognostic value.

GRADE 0 FOOT

A grade 0 feet has no open lesions but is an at risk foot. A large amount of callus under a metatarsal head may act foreign body and lead to an open but hidden lesion. So one must separate callus and see whether there is any ulcer underneath. If any ulcer is present it should be reclassified as Grade 1

Grade 0 feet with deformities such as intrinsic minus, hammer or claw toes, Charcot's joint or hallux valgus need purpose designed shoes. When patient with long standing diabetes with neuropathy and angiopathy notes such lesion they should be treated so as to prevent further complications. They should be instructed regarding daily inspection of foot especially between web spaces, head and metatarsals, heel of the foot and also areas of early abscess formation. The areas should be kept dry as moisture leads to break down of

epithelium and may lead to fungal infection. Physician should remove patient's shoes and stockings and examine the feet and web spaces in between toes each visit.

GRADE 01 LESION

This consists of superficial ulcer but with thickness skin loss. Usually these occur in plantar surface of toes of metatarsal heads. But kissing lesion occurs in between toes caused by over tight shoes. This is due to repeated pressure leading to ischemia. Thus mainstay of treatment is to release pressure from ulcerated area, surrounding callus removal and ulcer debridement until healthy granulation is seen. Saline irrigation is usually enough in these relatively clean superficial ulcers. If infection is present, a wound swab should be taken and antibiotic therapy with broad spectrum agents should be started immediately.

The most important part of treatment is to relieve pressure until lesion heals and fitting of specialized shoes.

GRADE 02 LESIONS

The ulcer is deep and often penetrates subcutaneous fat down to tendon or ligament, but without abscess or bony infection. These patients should be admitted to hospital and blood and ulcer cultures should be taken and foot X rayed

Deep infected ulcer need to be debrided in ward under anesthesia. After debridement deep ulcer should be packed with eusol in gauze wick to encourage healthy granulation tissue growth. Otherwise simple saline guaze is advised. Topical antibiotics are not useful.

GRADE 03 LESIONS

They have deep infection with cellulitis or abscess formation often with underlying osteomyelitis. In management surgery is often needed. Foot x ray, ulcer pus culture and sensitivity is must. Absent foot pulses, low ankle pressure and diffuse arterial disease suggest that lesion will not heal without amputation. Doppler studies may help to decide whether to persist with conservative treatment or proceed with local amputation. If the lesion is purely neuropathic, conservative treatment is sufficient since ulcer usually heals.

Initial treatment constitutes bed rest, elevation of foot, antibiotics according to culture and sensitivity. Patient with serious foot infection needs intravenous antibiotics, insulin therapy Grade 3 foot with good blood supply can be treated without amputation, with surgical drainage, dressing and wound irrigation. Amputation may be needed if severe infection or progressive anaerobic infection is present.

GRADE 04 LESIONS

Treatment is same as Grade 3 lesion. Avoid pressure bearing either with special shoes or bed rest is the mainstay of treatment.

When distal vascularity is adequate it is worthwhile trying conservative management. On the other hand, when there is gangrene of toes with absent pulses, a history of claudication or reduced Doppler pressure; healing is unlikely unless blood flow is improved. Arteriography is indicated to see whether bypass or angioplasty is indicated. If neither is possible, if there is no rest pain, then a period of conservative treatment is worthwhile. A painless black toe with dry gangrene often amputates spontaneously if left alone.

It is often difficult to decide when major amputation is required. In a previously mobile patient, a below knee amputation is better than above knee amputation because of better rehabilitation. But in elderly bedridden patient with peripheral gangrene and poor change of mobility an above knee amputation is required.

GRADE 05 LESIONS

These patients have extensive gangrene of the foot and needs urgent hospital admission, control of diabetes and infection and major amputation.

AMPUTATIONS IN DIABETIC FOOT

Amputation is believed to be one of the oldest surgical procedures that humanity has known, going back several thousand years at least to the times of Hippocrates. Ambrose Pare a French surgeon in the early 16th century is regarded as the father of amputation surgery.

Clinical assessment of the need of amputation includes Grade 5 and 4 lesions, severe sepsis with ketosis and absence of vascularity.

Final decision regarding level of amputation is taken as late as the skin incision. Supposing if the patient consented for below knee amputation and on cutting skin doesn't bleed then the patient should be informed about the possibility of above knee amputation and consent should be obtained for it.

SURGICAL TECHNIQUE

The gangrenous foot or leg is covered in a drape. Remainder of the limb is thoroughly cleansed with 10 minutes surgical wash followed by povidone iodine solution painting.

AMPUTATION OF TOES

Amputation of terminal phalanx of great toe

If functionally useful stump is to be obtained it is important to preserve the base of terminal phalanx into which the long flexor tendons and extensor tendon are attached. If possible take along plantar flap so that scar is not subjected to pressure.

Amputation through proximal phalanx of great toe

Stump containing less than one complete phalanx is to little or no value for weight bearing. Not more than the base of the phalanx should be therefore preserved.

Amputation of great toe at its base

The base of the proximal phalanx should be retained if possible so that insertion of the short muscles of the toe remains intact.

Disarticulation of lateral four toes:

Racquet approach is employed. Other techniques are similar.

Amputation of other toes

The toes are disarticulated at the metatarsophalangeal joint, the arteries secured and tendon allowed to retract.

Transmetatarsal amputations

This amputation is under taken using a long posterior flap, which extend to a level just proximal to the flexion crease at the base of the toes. Dorsal part of incision crosses the neck of metatarsals at which level bones are to divide.

The vascularity being in doubt the flaps are handled with most gentleness and not held with dissecting forceps. Sutures are kept at least for 14 days.

Transmetatarsal amputation (Lisfrank) level

This is performed through the tarsometatarsal joint and usually results in good partial amputations. Plantar incision should be slightly distal than dorsal incision whenever possible. Deeper tissues are incised to the bone. Tarsometatarsal joints are then disarticulated. The extensor and flexor tendons are sectioned at musculoskeletal junction whenever possible. The anterior tibial tendon is not disturbed in the amputation since its attachment to navicular bone is useful in dorsiflexion. Peroneus longus tendon, which is cut on plantar aspect, should ideally be reattached to navicular bone or one of the cuneiform on the medial side of the foot. Peroneus brevis muscle should be reattached to cuboid. The resulting partial foot amputation can be fitted with modified shoe.

Midtarsal amputation (Chopart amputation)

It is a disarticulation between the os calcis and cuboid bones and talus and navicular bones. If anterior tibial tendon is not attached to neck of talus, an equinus deformity is produced by unopposed action of gastrocnemius muscle. Because of this Chopart amputation is seldom used now.

SYME'S AMPUTATION

This operation first described by Syme's in 1842 is a classical amputation in the region of the ankle. The tibia and fibula are divided at or immediately above the level of ankle joint. The ends are covered with a single flap obtained from skin of heel. The end of stump is at a height of 6 to 8 cm from the ground.

Syme's amputation has a durable stump. 50 % of persons are able to walk with stump without prosthesis. Syme's amputation has a very good role in patients with diabetic foot. Wagner (1977) presented a good series of successful Syme's procedure in diabetic patients with foot ulcers.

MODIFIED SYME'S AMPUTATION

A modification suggested involves elliptical incision and division of tibia and fibula at a higher level. But this modification has nothing to commend and has not been approved widespread.

BELOW KNEE AMPUTATION

Amputation at the below knee level through middle third of leg is the operation of choice when it is not possible to conserve the foot and the heel. Ideal length of tibial stump is 14 cm. A stump shorter than 8 cm tends to slip out of the socket of the artificial limb and is very difficult to accommodate the patellar tendon bearing prosthesis. When it is not possible to save at least 8 cm

of stump it is better to go for disarticulation of knee joint or above knee amputation.

TECHNIQUE

In an ischemic limb amputation with long posterior flap is ideal. Posterior skin of skin is better perfused than the anterior skin. The skin incision is therefore transverse across the front of leg approximately half inch below the level of tibial transection. Incision is extended medially and laterally to mid leg and then it curves downwards to produce a long posterior flap which is equal to diameter of the leg at the level of amputation so that a free and tension less skin closure is possible.

Lateral portion of amputation is performed first dissecting through the anterior muscle compartment. The fibula is then exposed through extraperiosteal approach and transected approximately half an inch above expected level of tibial transaction. Then tibia is exposed extraperiosteally and divided half an inch above the level of skin incision. Anterior border of tibia should be beveled to prevent skin penetration. Posterior tibial vessels are identified and doubly ligated. Posterior calf musculature is then filleted down muscle should be dissected from deep fascia. The wound is closed in 2 layers with anterior and posterior fascia being approximated. A red rubber drain is placed near the site of closure and kept for atleast 24 hours.

After dressing, a plaster slab is applied keeping knee in 10 degree flexion. This helps to reduce edema in the limbs and thus facilitate sooner fitting of artificial prosthesis. It keeps the knee in optimal position and thus reduces flexion contractures. And suture removal is done after 14 days.

ABOVE KNEE AMPUTATION

Either long anterior skin flap or posterior skin flap is used. The length of flap should be two third of diameter of limb at the level of amputation. Level of bone section should be selected. Periosteum should not be stripped but on be cut. Nerves are directly identified and ligated and cut proximally. As much as length of stump should be preserved as possible. Drains are kept for 48 hours. Elastic compression bandages, which should be put above the waist region, are recommended. Patient can be fitted with preparatory lower limb prosthesis approximately 3 to 4 weeks after operation depending on healing of wound. Definitive prosthesis is usually put after two to four months.

MATERIALS AND METHODS

SOURCE OF DATA

This study was conducted at Government Rajaji Hospital, Madurai in the Department of General Surgery. 125 cases of diabetic foot admitted to the II

surgical unit in both male and female wards during the period of August 2003 to February 2006 were taken up for the study.

INCLUSION CRITERIA

All ulcers of the foot in patients who are suffering from diabetes mellitus are included in the study.

METHOD OF COLLECTION OF DATA

- Detailed history taking
- Thorough physical examination
- Investigation

RESULTS

TABLE 1 AGE FREQUENCY

Age group	No. of cases	Minor surgery	Major surgery	conservative	Percentage

20-29	3	0	0	3	2.4
30-39	8	1	2	5	6.4
40-49	24	6	2	16	19.2
50-59	28	15	4	11	22.4
60-69	42	5	7	16	33.6
70-79	18	5	5	10	14.4
79-80	2	0	0	2	1.6

TABLE 2 TYPE OF DIABETES

Type	No. of cases	Percentage
NIDDM	107	85.6
IDDM	18	14.4

TABLE 3 SEX RATIO

Sex	Total	Conservative	Surgical
Male	69	42	27

Female	56	40	16

TABLE 4 DURATION OF DIABETES

Duration of years	**No of case**	**Surgical**	**Conservative**	**Percentage**
Unknown	25	12	13	20
0-5	46	17	24	36.8
6-10	27	9	13	21.6
11-15	23	14	19	18.4
>16	4	-	4	3.2

TABLE 5 TREATMENT TAKEN

Modes of treatment	**No of cases**	**Percentage**

Diet control	12	9.6
Oral Hypoglycemic agents	50	40
Insulin	22	17.6
Insulin and oral hypoglycemics agents	16	12.8
No treatment	25	20

TABLE 6 INFECTIONS

Microorganisms	**No of cases**
Streptococci	7
Staphylococci	26
Pseudomonas	2
Klebsiella	1
E. Coli	4
Proteus	6
Mixed growth	12
No growth	10

TABLE 7 RESULT OF TREATMENT

Procedure	**No of cases**	**Discharged**	**Death**
Wound debridement	63	63	0

Split skin graft	9	9	0
Minor amputation	23	23	0
Major amputation	20	17	3
No treatment	10		10

TABLE 8 RISK FACTORS IN DIABETIC FOOT ULCERS

Risk factors	**No of cases**	**0**	**1**	**2**	**3**	**4**	**5**	**Percentage**
Neuropathy	29	0	1	10	2	7	9	23.2
Peripheral vascular disease	29	0	0	5	5	8	11	23.2
Foot deformity	6	0	0	4	0	1	1	4.8
Previous ulcer/amputaion	5	0	0	0	0	2	3	4
Vision/mobility impairment	0	0	0	0	0	0	0	0

TABLE 9 GRADING AT PRESENTATION

Grade	**Total cases**	**Conservative**	**Surgical**
0	0	0	0

1	44	44	0
2	30	18	11
3	22	1	16
4	16	0	13
5	13	O	9

TABLE – 10 AWARENESS OF THE DISEASE

	No	Percentage
Awareness of diabetes	100	80 %
Awareness of foot disorders and special foot wears.	7	5.6 %

ANALYSIS AND DISCUSSION

An analysis of 125 cases of diabetic foot ulcers was done. These cases were admitted and treated in II surgical unit in both male and female wards in

Government Rajaji Hospital, Madurai during the period of September 2003 to February 2006.

AGE

Peak age incidence occurred in 60 – 69 year group. Maximum number of amputations was also done in this age group. It is surprising to know that lesions occurred even in young people with diabetes mellitus. There were three such cases in 20 – 29 year group. All these Grade 1 or 2 lesions treated conservatively.

INCOME

Contrary to the fact that diabetic foot disorders is more prevalent in the affluent population, nearly 92% (115) patients in the study belonged to the poor socio economic group. This is similar to the reports from other studies in U.K 82%[6,41]. Nutritional status is not assessed in our study. But studies in Sweden[19] on nutritional supplementation had shown no advantages on healing of ulcer. The distribution of the diabetic foot ulcers is equal in all communities

SEX

Like many other diseases, males were affected more than females. There is slightly higher incidence of diabetes mellitus in males and male persons are more prone for diabetic foot ulcers due to trauma, bare foot walking, vascular disease and smoking etc. In our study, out of 125 cases males constituted 69

and females 56. Similarly male sex is associated with more incidence of amputation[41].

DURATION OF DIABETES

A significant number of patients were diagnosed to have diabetes only after admission. They had a high degree of lesions which needed surgical intervention (48%). Most of the patients had duration of up to 5 years which had the least incidence of high grade lesion which were treated by conservative management (52%). Only four patients had diabetes duration of more than 15 years and all of them were treated conservatively. Our study has no correlation between duration and the prognosis of disease. But studies from Oxford community study in U.K. shows that diabetes foot ulcer is directly related to the duration of diabetes mellitus[6,41].

MODE OF PREVIOUS TREATMENT

Nearly 32 % of the patient had irregular treatment for diabetes mellitus. Out of the 100 known diabetics 40% patient had oral drugs, 17% patient had insulin and 13 % had both insulin and oral hypoglycemic agents. As much as 20% had no treatment for diabetes previously.

INFECTIONS

Most of the pathogens isolated contained growth of Staphylococcus aureus. It constituted maximum number of cases (44 %). Mixed growth formed the next common group (20 %). Gram negative formed the other common organisms grown in culture. Staphylococcus[7] cultured in pus were resistant to almost all antibiotics, which reflects the general trend worldwide. MRSA and MRSE[7] were the common isolated in other studies in Manchester UK. Even though the studies in Germany have advised no antibiotics for uncomplicated trophic ulcer[5] we routinely treated with antibiotics. The gram negative organisms were commonly isolated in other study in Chennai[11]. The gram negative organisms were usually resistant to most common antibiotics and sensitive to amikacin. But many time the culture and report comes on an average of 5 days treatment is guided by and what the hospital strains are and the antibiotics are effective. It is true that proper surgical debridement, tissue oxygenation and wound dressing are more important than the antibiotic usage.

ASSOCIATED CARDIOVASCULAR DISEASE

44 percent of the diabetic foot ulcer patients had associated coronary artery disease. The association was stronger with male sex 63% which indicates their inherent susceptibility towards coronary disease.

NEUROPATHIC LESIONS

In the present study 29 patients were found to have neuropathy. The patient with neuropathy varied from 35 – 80 year old, majority had a history of diabetes of more than five years. This shows that the peripheral neuropathy is common in long standing diabetic patients.

	Root	Grams	Duncan	Pennsylvania	Present study
	1985	1969	1969	1969	2006
No of cases	3175	264	354	614	125
Neuropathy	1206	84	125	175	29
Per	37.9	31.8	35.3	28.5	23.2

ISCHEMIC LESIONS

Table Incidence of Gangrene

	Pennsylvania	Present study
No of cases	614	125
Gangrene	274	29
Percentage	44.7	23.2

44.8 percentage of ischemic lesions seen in Pennsylvania hospital was compared with the present series which is 23.2 %. This variation is probably due to more number of Wagner type I and 2 treated. The vascular status of the patient is directly related to the salvage status of the limb. .

MANAGEMENT

All patients were treated under the surgical team comprising of diabetologist, vascular surgeons, plastic surgeons and other specialists. The team approach has important role in managing the ulcers. Studies in Italy and Boston have shown that the surgeon controlled team healed the lesions better than diabetologist[15,17,18]. All patients were put on injection insulin, with dose adjusted according to the level of control. 17 patients were admitted with Ketoacidosis, which was managed by intravenous fluids and insulin. The requirement of insulin does not correlated with the severity of the ulcer grade and ranged from 3units of human recombinant insulin to 75 units. But other studies Portland Oregon has showed that multiple daily insulins or intravenous insulin infusion therapy has improved the wound healing and less mortality. Similarly hyperglycemia is associated with poor healing

DURATION OF STAY

The mean stay in the hospital was 18 days and it was more 31 days for the patients who had major amputation. The cost of treating a diabetic foot ulcer is a huge burden to the health care as it is time consuming and costly. The duration is 7 days and four times if amputation occurs as per the analysis of Apelqvist et al 1995 in U.S.A. In German population the average duration of hospital stay is 92 days and in other centers of India it is 118 days. The relative

shorter duration in our set up may be due to the fact that early decision regarding amputation, poor hospital resources (bed strength) causing early discharge. This show the burden of the diabetic patients in our health set up. Another fact is that more than 16 percentage of patients were admitted in emergency basis with pain, gangrene, septicemia and ketoacidosis.

ANTIBIOTICS

In our institution, a broad spectrum antibiotics were administered at the time of admission, after taking swab for culture and sensitivity. Later when the report was ready the antibiotics were changed accordingly. Ampicillin and Cefotaxime along with Metronidazole were administered parenterally and later changed after pus culture and sensitivity report was available. The antibiotics were continued for seven to ten. No topical ointment was used except EUSOL dressing for which the patients responded well.

TREATMENT

55.4 % patients studied were managed conservatively. They were treated with daily dressing and wound debridement etc. These patients constituted cellulitis, trophic ulcers and abscess. Daily eusol cleaning and dressing is efficient along with proper wound debridement in our patients. The other modalities like Datkin's solution. enzymatic preparations, biosurgical dressings moist dressings, hyperbaric, dermal replacement (dermagraft) composite

replacement (apligraft) are the other older and newer dressing methods and none have been shown to be better in terms of cost , efficiency and cure.

32 patients underwent minor procedures one are two toes amputations or Split Skin Graft etc. SSG was done in 9 patients and had a high rate of failure. Only 5 patients had good take. 23 patients had minor amputation of one or two toes.

21 patients had major amputation in the form of below knee amputation 19 and above knee amputation in 4 patients

MORTALITY

Mortality was 13 deaths out of which 3 were postoperative deaths. The most common cause of death was septicemia (7) coronary events (2) major cerebrovascular accidents (1). 9 Patients who died had diabetic ketoacidosis. Similar to other study in Dutch population the mortality was related to the Wagner grade, 4 and 5 were associated with higher mortality[4].

RISK FACTORS

Presence of previous amputation was 4 % in the overall patients and was 10 % in the patient who underwent amputation during the course of illness. One of our patients had multiple amputations. He had two toes amputated sequentially during a period of admission and later followed by below knee

amputation of the same limb 6 month back. 4 year back he was admitted for gangrenous foot with septicaemia requiring below knee amputation of the other limb.

Presence of foot deformity is less in our patients like hammer toe 3, and hallux valgus 5, and Charcot's joint 1 case, when compared with western studies which says that 24 percent of the foot ulcers are preceded by a deformity. The other deformities described in their literature include hammer toe, claw toe, hallux deformity, pes planus, pes cavus and Charcot foot.

Risk factors	***No of cases***	***0***	***1***	***2***	***3***	***4***	***5***	***Percentage***
Neuropathy	***29***	***0***	***1***	***10***	***2***	***7***	***9***	***23.2***
Peripheral vascular disease	***29***	***0***	***0***	***3***	***5***	***8***	***13***	***23.2***
Foot deformity	***6***	***0***	***0***	***4***	***0***	***1***	***1***	***4.8***
Previous ulcer/amputation	***5***	***0***	***0***	***0***	***0***	***2***	***3***	***4***
Vision/mobility impairment	***0***	***0***	***0***	***0***	***0***	***0***	***0***	***0***

None of the patient were vision impaired to blindness as per as WHO criteria[20].

When compared with the risk factors along with the grading of the ulcers it is clear that more the number of risk factors more the grade of the ulcer.

In Grade 5 lesions neuropathy is present in 69 %, peripheral vascular disease in 100% and previous ulcer or amputation in 23% and foot deformity in 8% of the patients.

In Grade 4 lesion neuropathy is present in 43.5 %, peripheral vascular disease in 50 % and previous ulcer or amputation in 6 % and foot deformity in 13 % of the patients

In Grade 3 lesion neuropathy is present in 1 %, peripheral vascular disease in 5 % and previous ulcer or amputation and foot deformity in none of the patients

In Grade 2 lesions neuropathy is present in 33 %, peripheral vascular disease in 11%, foot deformity in 4% and previous ulcer or amputation none of the patients.+

In Grade 1 lesions neuropathy is present in one of the patient and other patients had no risk factors.

Other studies have shown that neuropathy is commonest risk factor for ulcers and amputation. But our study shows neuropathy and vascular disease are both risk factors for amputation 23%.

Only 7 of the patients had the awareness of specialized foot wear for diabetics and all of them had Grade 1 Wagner foot on treatment.

As Wagner pointed arterial blood flow is an important predictor of survival of the limb, showing that the all patients with vascular impairment had amputation of limbs or toes[3].

AMPUTATION

The rate of amputation is less than 5% in USA and 30 % in Vietnam[43] In India[13] it is about 18 %. Our study shows rate of amputation of about 32 % which is very high. The risk factor, peripheral vascular disease was important factor as in other studies[17,12]. The poor socioeconomic status (92 %), late presentation, lack of adequate treatment (33%) and lack of adequate knowledge of diabetic foot care(20%) may be the reason for these high rates.

REHABILITATION.

14 Patients who had above knee amputation had crutches at the 10th post operative day and were provided below knee prosthesis after healing of wounds. 7 patients had changed their job and are able to rehabilitate. All the above knee amputees were disabled and were not able to lead their normal life.

Even in advanced countries like Sweden only 53 % could be able to get proper prosthesis in above knee amputation and 46% died within 2 years[9]. Diabetic foot ulcers causes a adverse impact on the quality of life[16].

AWARENESS.

The public as found does not know about diabetes and its complications from the study. As high as 25 patients had no previous treatment for diabetes mellitus (20%). Only 7 patients out of 125 are aware of the specialized foot wear for diabetic foot lesions. But public awareness programs or special programs for GPs in Algeria from 1994 had not improved the outcome of diabetic foot lesions[18.] . But to achieve the St Vincent's declaration[10] of preventing the amputation, the Sweden study shows proper education and awareness programmes are necessary to the high risk people[8].

CONCLUSION

Majority of the foot ulcers in diabetic patients occurred in poor socioeconomic patients. . Peak age of occurrence of the disease is 60 to 69 and males have high risk of foot lesions.

Ignorance of the disease and lack of proper awareness of the foot lesions are important risk factors of the disease

Duration of diabetes is not directly related to the disease in our study but inadequate treatment and no treatment were associated with major amputations.

Both peripheral vascular disease and neuropathy was equally present in the patients with diabetic foot ulcers and are the commonest risk factors.

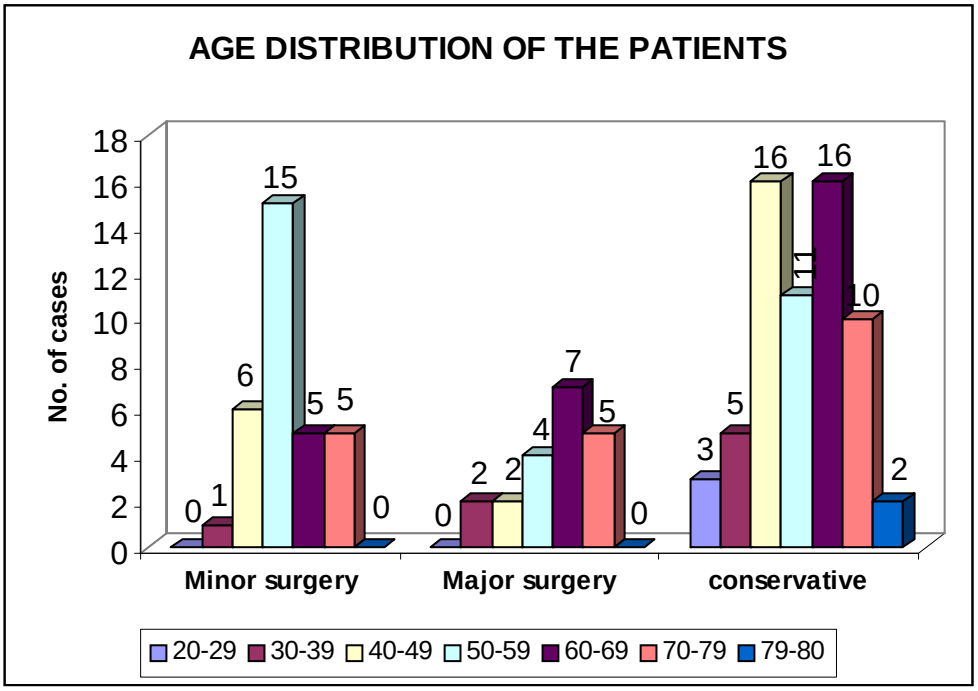
Most common isolate in bacterial culture is Staphylococcus aureus.

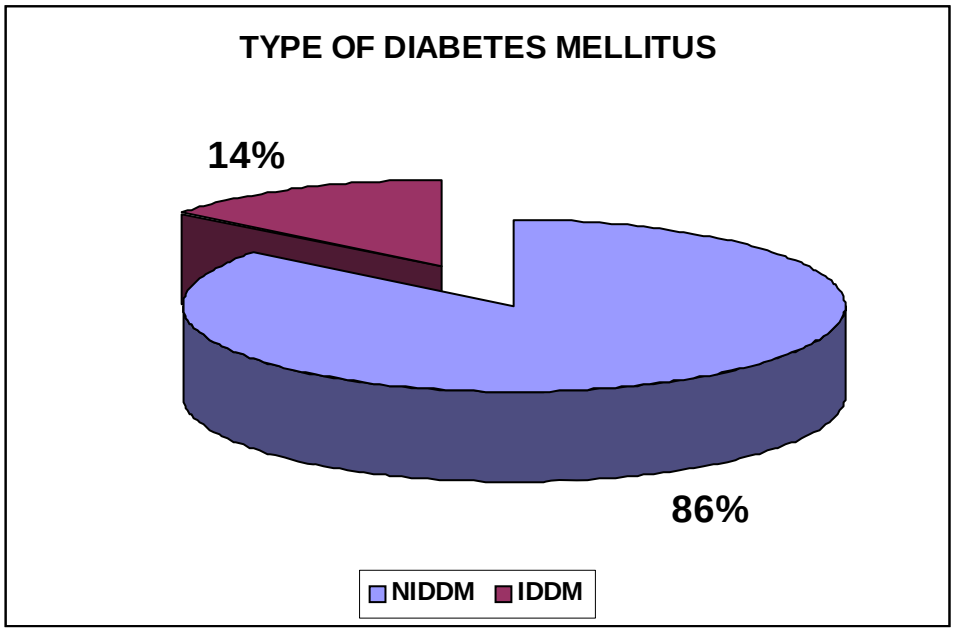
The most important prognostic factor for major amputation is Wagner staging.

Diabetic foot ulcers is the important cause for prolonged hospital stay, the mean stay is 18 days and adversely affects the quality of life.

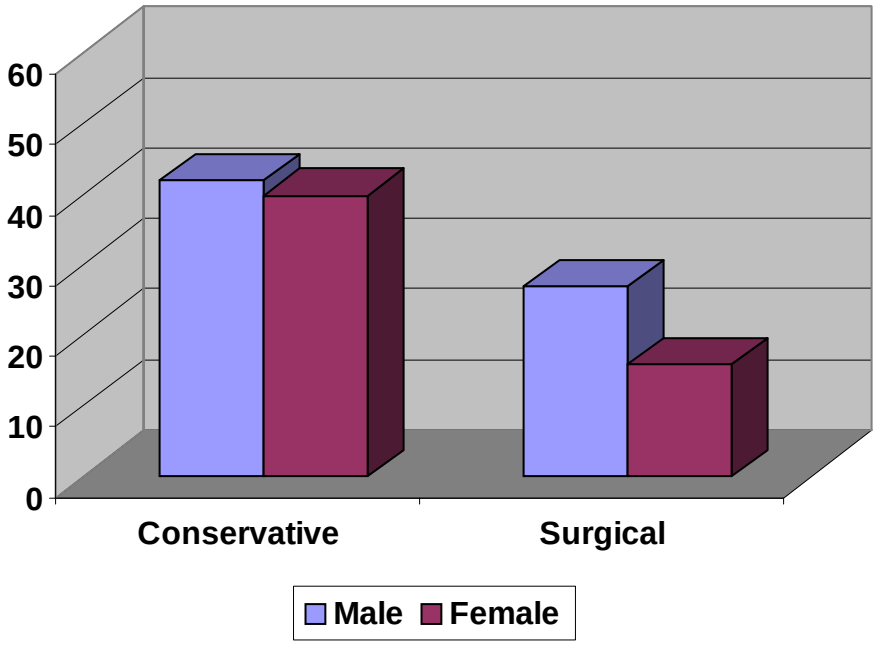
The percentage of amputation in diabetic foot ulceration is 32%, which is very high, and the mortality is 10%.

For achieving the St Vincent's declaration which calls for decreasing the rate of amputation proper awareness programmes are necessary.

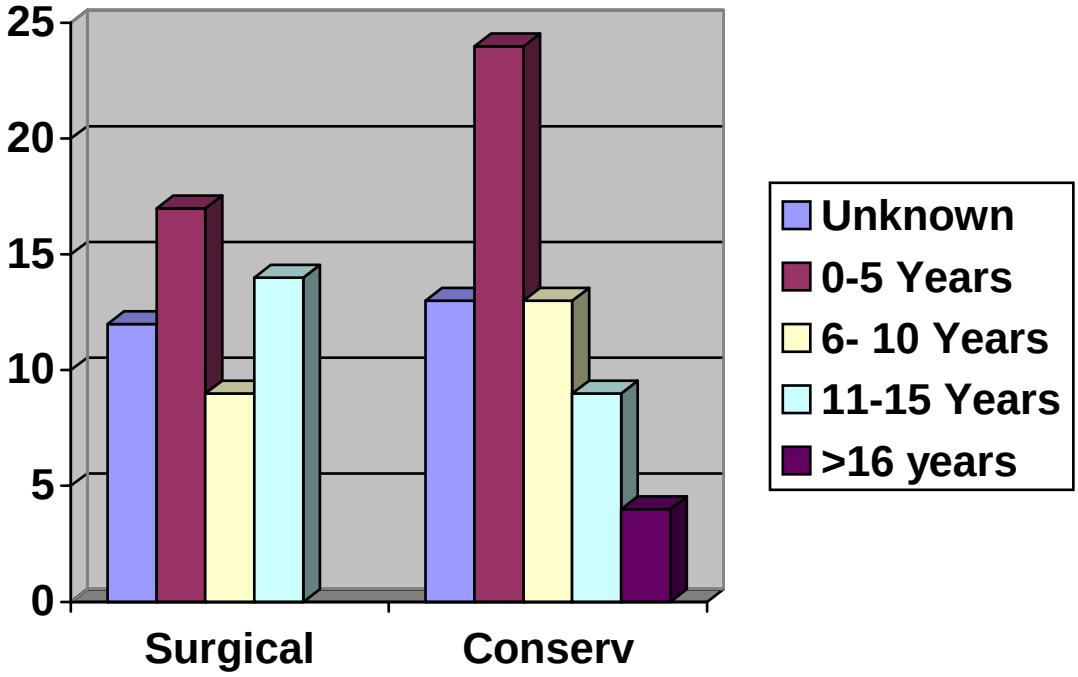

AGE DISTRIBUTION OF THE PATIENTS
No. of cases
18
16
14
12
10
8
6
4
2
0
0 1 6 15 5 5 0
0 2 2 4 7 5 0
3 5 16 11 16 10 2
Minor surgery
Major surgery
conservative
20-29
30-39
40-49
50-59
60-69
70-79
79-80


TYPE OF DIABETES MELLITUS
14%
86%
NIDDM
IDDM

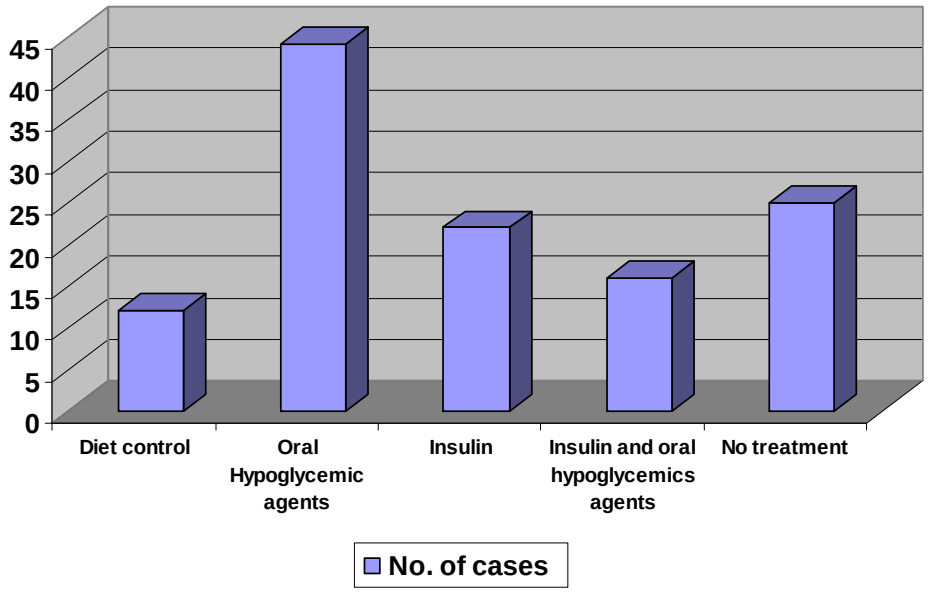
SEX DISTRIBUTION



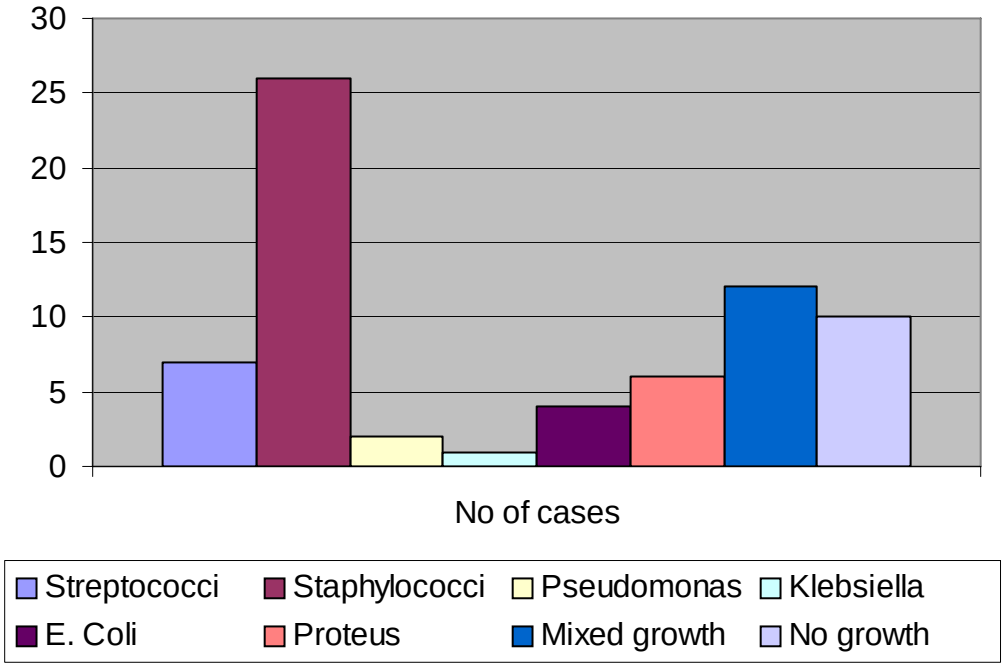
DURATION OF DIABETES MELLITUS



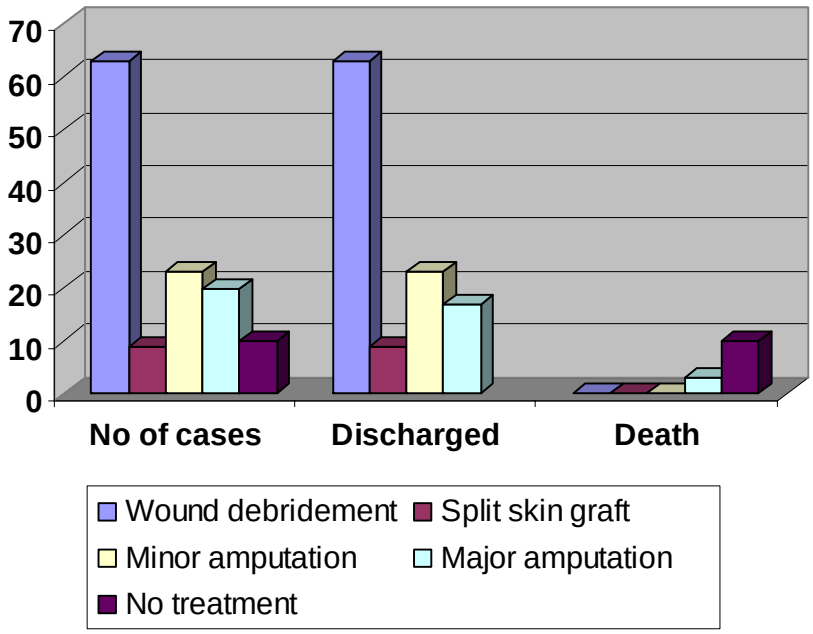
MODE OF MANAGEMENT



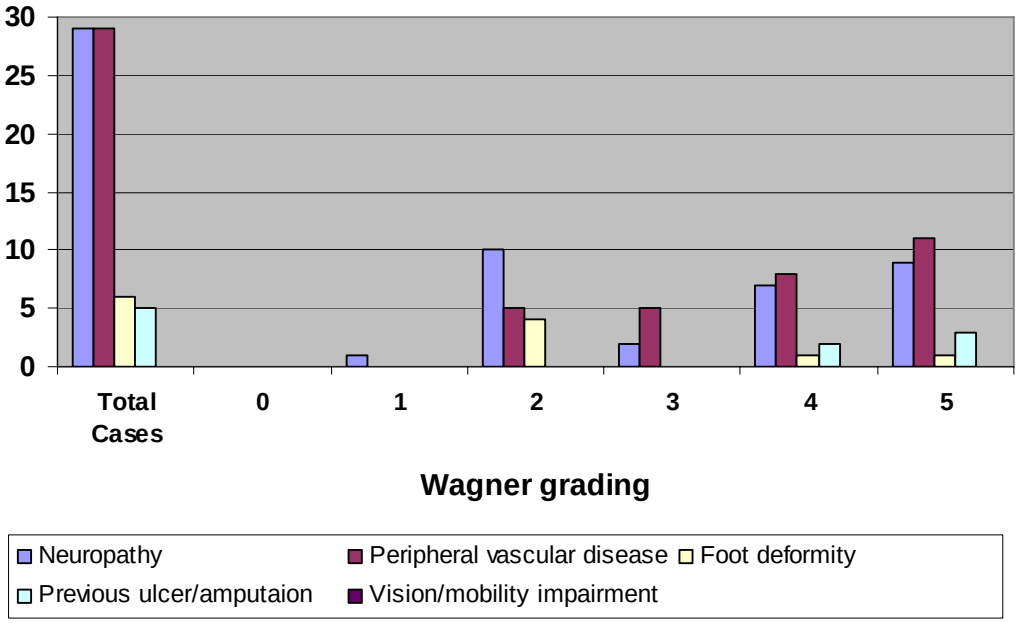
MICRO ORGANISMS CULTURED



MODES OF TREATMENT



RISK FACTORS FOR WAGNER GRADING


30
25
20
15
10
5
0
Total Cases
0
1
2
3
4
5
Wagner grading
Neuropathy
Peripheral vascular disease
Foot deformity
Previous ulcer/amputaion
Vision/mobility impairment

DOPPLER ASSESSMENT OF VASCULAR STATUS

SENSORY TESTING WITH MONOFILAMENT

NEUROPATHIC TROPHIC ULCER

DIABETIC CELLULITIS WITH DEEP SEATED ABSCESS

DIABETIC FOOT DRY GANGRENE

DRY GANGRENE AFTER ABOVE KNEE AMPUTATION

RAW AREA FOREFOOT READY FOR SSG

SSG DONE

PROFORMA

Serial number:

Name: IP number: Age:

Sex: DOA:

Occupation: DOS:

Address: DOD:

Presenting Complaint

Risk factors: Deformities, previous ulcer, previous amputation sensation, and vision/mobility impairment.

Past History

Know Diabetic: Y/N

Type of Diabetes: NIDDM / IDDM

Duration: YRS MONTHS

Method of control Nil/ Diet / oral drugs / insulin

History of DKA : Y/N once / many times

History of admission for diabetic complication (Type of complication)

History of disease of the CVS Y/N

History of peripheral Vasculopathy Y/N

History of peripheral neuropathy Y/N

History of foot ulcers Y/N

History of amputation Y/N

Personal History

Smoker / Alcoholic Duration

History of Prolonged drug intake Y/N

General examination

Pallor/ Icterus / Clubbing / Cyanosis / edema of feet

Pulse rate PR

Systemic Examination

CVS RS

GIT CNS

URINARY RETINOPATHY

Peripheral Vascular involvement Y/N

Peripheral Neuropathy Y/N

INVESTIGATIONS

Blood

Hb TC DC ESR

Initial Blood Sugar Repeat values

Fasting Blood sugar

Post postprandial blood sugar

Blood Urea Serum Creatinine

Urine Albumin Y/N Sugar Y/N Acetone Y/N

Plain X ray of foot for Bone involvement

Pus C/S

Organism

Sensitivity

Doppler Study of both lower limbs

Course in the hospital

Adequate control of DM Y/N

Insulin dose for adequate control:

Time taken for control

In Hospital complication and management if any

Management of Foot

Conservative/ Surgery

Surgery Split skin graft / amputation

Type of amputation: Minor / Major

Nature of amputation

Follow up

Functional impairment mild / moderate / severe

Change of Job Y/N

Awarness of diabetes and its treatment Y/N

Awarness of specalised food wear Y/N

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